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# Health and Environmental Effects Profile for Alachlor

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# HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR ALACHLOR

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16. ABSTRACT

The Health and Environmental Effects Profile for alachlor was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Alachlor has been evaluated as a carcinogen. The human carcinogen potency factor ($q_1^*$) for alachlor is $1 \times 10^{-2}$ (mg/kg bw/day)$^{-2}$ for oral exposure.

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## PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched."

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or $q_1^*$ is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

## ACKNOWLEDGEMENTS

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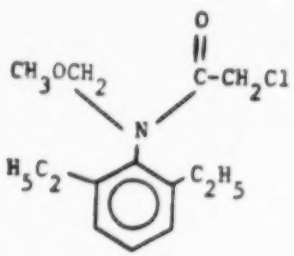
## LIST OF ABBREVIATIONS

| | |
|---|---|
| ADI | Acceptable daily intake |
| DNA | Deoxyribonucleic acid |
| $LC_{50}$ | Concentration lethal to 50% of recipients |
| $LD_{50}$ | Dose lethal to 50% of recipients |
| ppm | Parts per million |
| UV | Ultraviolet |

# 1. INTRODUCTION

## 1.1. STRUCTURE AND CAS NUMBER

Alachlor is also known as Lasso®, Lazo, CP 50144 and 2-chloro-2',6'-diethyl-N-(methoxymethyl)acetanilide (Von Rumker et al., 1974). The structure of alachlor is given below:



Molecular formula: $C_{14}H_{20}ClNO_2$

Molecular weight: 269.8

The Chemical Abstracts Service (CAS) Number for this compound is 15972-60-8.

## 1.2. PHYSICAL AND CHEMICAL PROPERTIES

Alachlor is a nonvolatile crystalline solid; some of its physical properties are listed below:

| | |
|---|---|
| Melting Point: | 40°C (Von Rumker et al., 1974) |
| Density: | $d_{15.6}^{25}$ 1.133 (Martin and Worthing, 1977) |
| Water Solubility: | 240 mg/ℓ at 23°C (Martin and Worthing, 1977) |
| Vapor Pressure: | $2.2 \times 10^{-5}$ mm Hg at 24°C (Beestman and Deming, 1974) |

## 1.3. PRODUCTION DATA

Alachlor is an α-chloroacetanilide herbicide manufactured by Monsanto in Luling, LA, and Muscatine, IA. Production data for 1982 were not available, but SRI (1976) estimated that 126 million pounds were produced in 1976. It is synthesized by reacting chloroacetyl chloride and 2,6-diethyl-N-methoxymethyl aniline in methanol (SRI, 1976).

## 1.4. USE DATA

Alachlor was patented by Monsanto in 1966 (USP 3,442,945). It is a wide spectrum preemergence or early postemergence herbicide used to control annual grasses and many broad-leaved weeds in such crops as corn, soybeans, peanuts, cotton, sugar cane, radish, rapeseed and other brassicas. It is applied at ~2 kg/ha, and has residual action lasting ~10-12 weeks (Martin and Worthing, 1977). Alachlor is distributed in two types of formulations; a 60% emulsifiable concentrate, or a granular form that contains 40% alachlor (SRI, 1976).

## 2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

Specific data regarding the fate of alachlor in the atmosphere were not located in the available literature. Alachlor may enter the atmosphere as an aerosol from spraying, or to a lesser extent from wet exposed soil under windy conditions (Beestman and Deming, 1974). Although the vapor pressure of alachlor is low, Hargrove and Merkle (1971) and Beestman and Deming (1974) reported significant losses caused by volatilization from moist silt and sandy loam soils, respectively. Atmospheric photodecomposition of alachlor may occur, since it has been demonstrated to occur by sunlight on soil thin layers (Fang, 1977, 1979).

Many investigators have evaluated the fate of alachlor in soil and water, as it is directly applied to soil before the emergence of crops. The dissipation of alachlor from soil is controlled primarily by microbial decomposition, but soil type, temperature, pH and other factors also influence dissipation. Microbial decomposition has been estimated to account for 90% of the total loss from soil (WSSA, 1979).

In a laboratory assay, Beestman and Deming (1974) applied alachlor (~2.2 kg/ha) to sterile (autoclaved) and viable silt soil, and observed the half-lives for dissipation of the compound to be 470 and 8 days, respectively. In field experiments, alachlor applied to Ray silt (pH 6.7) and Wabash silty clay (pH 7.0) had half-lives of 4.0 and 7.3 days, respectively. The authors concluded that the soil microorganisms contained in these soils rapidly degraded alachlor under laboratory and field conditions. The rate of dissipation was observed to follow first order kinetics.

The formation of 2-chloro-2',6'-diethylacetanilide when alachlor was incubated with sandy loam soil was reported by Hargrove and Merkle (1971). This study also demonstrated the formation of this degradation product when

alachlor was reacted with 5N aqueous HCl at 40°C for 72 hours. It is unclear whether the product resulted from microbial degradation when incubated with soil or by chemical reaction, since the soil was acidic (pH 4.9).

The fungal metabolism of alachlor was further investigated by Tiedje and Hagedorn (1975), who observed four metabolites resulting from 7-day incubations with Chaetomium globosum. Uniformly ($^{14}C$) ring-labeled alachlor was incubated with each of eight species of soil fungi; only C. globosum completely degraded the compound. No $^{14}CO_2$ was observed to be released during incubations; 68% of the chlorine was released as chloride. The major metabolite was 2,6-diethylaniline; smaller amounts of 2-chloro-2',6'-diethylacetanilide, 2,6-diethyl-N-(methoxymethyl)aniline and 1-chloroacetyl-2,3-dihydro-7-ethylindole also were formed.

Chahal et al. (1976) reported on the degradation of alachlor by two species of soil fungi, Penicillium sp. and Trichoderma sp. Both species were observed to generate two metabolites after 30 days of incubation with alachlor as the sole source of carbon. The metabolites were identified by their Rf values on thin layer chromatographic plates. Fungal decomposition of alachlor by Rhizoctonia solani was reported by Smith and Phillips (1975). The fungus was incapable of utilizing alachlor as the sole carbon source, but it effectively degraded the compound ($t_{1/2}$ ~4 days) when in the presence of 17.6 mM sucrose.

Kaufman and Blake (1973) incubated alachlor with the soil fungus, Fusarium oxysporum, and reported the release of 22% of the chlorine over a period of 20 days.

Yu et al. (1975) reported the fate of alachlor in a model ecosystem. Ring-labeled ($^{14}C$) alachlor (13.5 mg) was placed in the "land" portion of an aquarium containing sand, water, sorghum, algae, Daphnia, Elodea, crabs,

fish, snails and mosquitos. The radioactivity in the various entities of the ecosystem was assayed up to 33 days later. Unchanged alachlor was not found in any of the organisms, but the water portion of the ecosystem contained ~1.8% of the parent alachlor. Eight degradation products were found in the water, and aqueous radioactivity increased from day 0 to day 5, then leveled off. Total radioactivity was higher in organisms than in water, but there was no magnification of radioactivity in food chains, since the radioactivity in algae → mosquito → fish, went from 0.66 → 0.23 ppm alachlor.

The soil affinity of alachlor was investigated by Wu (1980). Soil core samples from a Chesapeake Bay watershed sprayed with alachlor (~2.3 kg/ha) were taken over 148 days and analyzed for the compound at various depths. Alachlor remained in the 0-8 cm zone, and concentrations in deeper zones were <0.01 µg/g. This low leachability results from alachlor's tendency to adsorb to soil organic matter. About 0.2% of the applied alachlor was lost through surface water runoff; the flow-weighted mean concentation was 0.6 µg/ℓ. A soil sorption coefficient of ~200 for alachlor was reported by Kenaga (1980), indicating that some mobility of the compound may occur in soil. Weidner (1974) and Weber and Peter (1982) have also reported better adsorption of alachlor to soils with higher organic matter content. Further, Weber and Peter (1982) reported that soils with higher clay content tended to promote the adsorption of alachlor.

Minimal leaching of alachlor has been reported by Hayden and Smith (1980) in Saskatchewan sandy loam, clay and silty clay. Negligible amounts of alachlor were observed in the 5-10 cm level of soils 17 months after spray application of 2.5 kg alachlor/ha. These results are in agreement with those of Beestman and Deming (1974), who observed that <1% of the alachlor applied to soil in field test cores was found below the 4 cm level.

Baker et al. (1979) reported that 6 weeks after alachlor was applied (~2.4 kg/ha) to an Iowa watershed (mostly silt loam soil), ~0.1 and 1.0 ppm of its residues were found in water and sediment from surface runoff events. The authors estimated that runoff accounted for 0.1-0.48% of the alachlor applied.

The photodecomposition of alachlor on soil thin layers by sunlight was investigated by Fang (1977, 1979). Seven products were isolated, and were generated in greater amounts when in the presence of lower pH and higher soil organic matter content.

Tanaka et al. (1981) irradiated 100 ppm aqueous solutions of alachlor with 300 nm UV light for 135 minutes in the presence of surfactants. Alachlor losses were 1, 1 and 85% in solutions containing water only, Tergitol TMN-10 surfactant and Triton X-100 surfactant, respectively. This may be of consequence, since ~60% of the alachlor used is in the form of an emulsifiable concentrate, and may be more susceptible to photodegradation.

Alachlor is not expected to bioaccumulate to a large degree based on the study of Yu et al. (1975), wherein no parent alachlor was found in organisms and alachlor degradation products did not concentrate in a typical food chain. Kenaga (1980) predicted bioconcentration factors for alachlor of 28 and 9, respectively, based on its water solubility and soil adsorption coefficient. These low values also indicate that alachlor will not bioaccumulate.

## 3. EXPOSURE

Cabbage grown in soil sprayed with alachlor at the proposed recommended rate (3.4 kg/ha) and at twice this rate had no detectable residues when harvested ~6 weeks later (Hunt et al., 1980).

Brookhart and Johnson (1977) analyzed tissue samples from fish caught in nine rivers that empty into Chesapeake Bay. Alachlor was determined with gas liquid chromatography; all samples contained <0.1 ppm. Dudley and Karr (1980) reported no detectable alachlor in fish (creek chub) or water from various locations in the Black Creek watershed in Allen County, IN, in July 1977.

Some transport of alachlor must occur in the environment, as Spalding et al. (1980) reported detecting the compound in 2 of 14 groundwater samples from heavily sprayed land in Nebraska during the 1978 irrigation season. Well depths were 20-23 m, and alachlor concentrations of 0.071 and 0.018 µg/ℓ were observed in groundwater below medium textured silty loam and silty clay loam soils, respectively. The organic matter content of these soils was not reported.

Keith et al. (1976) detected alachlor (0.17-2.9 µg/ℓ) in some samples of finished drinking water from a New Orleans drinking water plant using the Mississippi River as a source. Konasewich et al. (1978) measured alachlor levels in Lake Erie water, and found concentrations of 0.08, 0.07 and 9.0 µg/ℓ in three samples.

Leung et al. (1982) evaluated the accumulation of alachlor upstream, within, and downstream of a new reservoir on the Des Moines River over a period of 14 months. The watershed upstream of the reservoir was 79% cropland. Alachlor concentrations were found to be highest in the summer months

at all three sampling locations. The mean concentrations were 115 (range = 0-816), 80 (range = 0-410) and 72 (range = 0-370) ng alachlor/ℓ of water for the upstream, reservoir and downstream sites, respectively. The authors estimated that 251 kg of alachlor was deposited during the study period (September, 1977-November, 1978).

## 4. COMPOUND DISPOSITION AND PHARMACOKINETICS

Information regarding the absorption, distribution, metabolism and excretion of alachlor in mammals was not found in the available literature.

Leavitt and Penner (1979) investigated the nonenzymatic reaction of [$^{14}$C] alachlor with a number of cellular thiol-containing compounds. They reported a pH-dependent reaction of alachlor with reduced glutathione, cysteine, dithiothreitol and coenzyme A. Even though these reactions produced a low yield at physiological pH, these data suggest that conjugation to thiols, catalyzed by the glutathione-S-transferases or similar enzyme systems, may represent an important detoxification pathway for alachlor.

## 5. EFFECTS

### 5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of alachlor were not found in the available literature. Alachlor is not scheduled for testing by the National Toxicology Program Carcinogenesis Testing Program (NTP, 1983).

### 5.2. MUTAGENICITY

Eisenbeis et al. (1981) tested alachlor and the most widely used combinations of alachlor and other herbicides in the Ames Salmonella mutagenicity assay. The herbicide and herbicide mixtures were tested, as the full strength compounds and as a diluted water mixture approximating a tank mix at the recommended concentration for a delivery rate of 15 gal/acre, in a spot test system using Salmonella strains TA1535, 1537, 1538, 98, and 100. Negative results were obtained in all strains, with all herbicide combinations, both with and without rat liver S-9 activation.

Wildeman and Nazar (1982) measured the mutagenicity of alachlor (50 µg/plate) in the Salmonella bioassay (strains TA98 and 100) with both rat liver S-9 and plant homogenates as the activating system. Alachlor treatment did not result in a significant increase in revertants using either activation system.

Probst and Hill (1980) and Probst et al. (1981) reported a strong correlation between the induction of unscheduled DNA synthesis (UDS) in primary rat hepatocytes and mutagenicity in the gradient plate assay (spot test) modification of the Ames Salmonella bioassay. UDS was measured by autoradiography, based on methyl-$^{3}$H-thymidine incorporation into the cellular DNA. The alachlor concentration selected for quantification of UDS was the highest concentration that did not produce cytotoxicity. Alachlor produced negative results in both the Ames assay and the induction of UDS.

Georgian et al. (1983) examined the effect of alachlor on human lymphocytes obtained from healthy volunteers (number unspecified). During the last 24 hours of incubation, 72-hour blood cultures were exposed to 1, 2, 4, 10, 20 or 40 µg alachlor/mℓ. Fifty metaphases were scored for chromosomal aberrations from each blood culture. There was a dose-dependent increase in chromosomal aberrations, with a concentration of 40 µg/mℓ producing a high level of cells with despiralized, shattered and concomitantly separated chromatids. No effects were observed at concentrations of 1 or 2 µg/mℓ.

Georgian et al. (1983) also tested alachlor for cytogenetic effects in vivo. Doses of 1.25, 2.50 or 5 µg alachlor/g bw were administered to groups of 2-3 Wistar rats by intraperitoneal injection, and bone-marrow cells were gathered 24 hours later. Fifty metaphases were scored from each animal. All animals in the high-dose group died within 2-4 hours of treatment and were, therefore, not examined. Doses of 1.25 and 2.50 µg/g bw produced a dose-related increase in total aberrant cells, breaks and gaps. In concurrent studies in which a group of 4 rats were fed diets containing 200 ppm alachlor (~1.7 µg/g bw) for 280 days, the incidence of chromosomal aberrations was not significantly increased over the controls.

Murnik (1976) tested a number of widely used herbicides in the Drosophila melanogaster dominant lethal assay. Alachlor (concentration not given) did not significantly increase the rate of apparent dominant lethals or the frequency of sex-linked recessive lethals.

Gentile et al. (1977) tested alachlor for mutagenicity in the yeast, Saccharomyces cerevisiae strain D4, using a plant-activation bioassay. In this assay, log phase yeast cell cultures are treated with an extract from maize plants exposed to known concentrations of herbicide. Dose-dependent

increases in mutations of ~2.6- and 3.3-fold were observed in assays using preparations from plants exposed to 10 and 25 ppm alachlor, respectively. Alachlor did not induce mutations, however, when applied directly to the yeast cells at a concentration of 1000 ppm.

Singh et al. (1979) found alachlor to be more mutagenic than N-methyl-N-nitro-N-nitrosoguanidine (MNNG), a powerful bacterial mutagen, in the blue-green alga, Nostoc muscorum. The significance of this observation to mammalian systems is questionable, since Nostoc muscorum would be expected to metabolize alachlor in a manner different from animal systems.

## 5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of alachlor were not found in the available literature.

## 5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of alachlor were not found in the available literature.

## 5.5. CHRONIC AND SUBCHRONIC TOXICITY

Pertinent data regarding the chronic or subchronic toxicity of alachlor were not found in the available literature.

## 5.6. OTHER RELEVANT INFORMATION

The Pesticide Manual (Martin and Worthing, 1977) classifies alachlor as a nonirritant with an $LD_{50}$ for dermal application of >2000 mg/kg (species not specified). NIOSH (1983) reports a dermal $LD_{50}$ of 3500 mg/kg for the rabbit. Acute oral $LD_{50}$s ranging from 462-3000 mg/kg have been reported (NIOSH, 1983), depending on the species being tested, as shown in Table 5-1.

TABLE 5-1

Acute Toxicity of Alachlor*

| Species | Route | $LD_{50}$ |
|---|---|---|
| Rat | oral | 1200 mg/kg |
| Mouse | oral | 462 mg/kg |
| Rabbit | dermal | 3500 mg/kg |
| Unknown mammal | oral | 3000 mg/kg |
| Unknown mammal | unknown | 1200 mg/kg |

*Source: NIOSH, 1983

## 6. AQUATIC TOXICITY

### 6.1. ACUTE TOXICITY

The 96-hour median lethal concentration ($LC_{50}$) of alachlor to rainbow trout, Salmo gairdneri, was 2.4 mg/ℓ (Johnson and Finley, 1980). The bluegill sunfish, Lepomis macrochirus, was more resistant, having a 96-hour $LC_{50}$ value of 4.3 mg/ℓ.

Data from an abstract of a Czecheslovakian study indicate that alachlor at concentrations of ~0.01 mg/ℓ showed minimal signs of toxicity in the goldfish, Carassius carassius, the guppy, Poecilia reticulata, the tench, Tinca tinca, and Leucaspuis delineatus (Jirasek et al., 1980). The carp, Cyprinus carpio, was more resistant, showing no toxic effects at the same levels (0.01 mg/ℓ). In abstracts of two Bulgarian publications, Doicheva (1978, 1979) reported that $LC_{50}$s of alachlor for fingerling carp, C. carpio, were 10.75, 7.14 and 4.67 mg/ℓ for 24-, 48- and 96-hour exposures, respectively. The maximum concentration showing no toxic effects for carp was 0.944 mg alachlor/ℓ (Doicheva, 1979). Adamek et al., 1981 reported that Carp exposed to alachlor at 0.006 and 0.009 mg/ℓ survived only 19-29 days. During this period, fish growth was inhibited. Survival and growth rate of unexposed (control) fish were not available from the abstract of this study (Adamek et al., 1981).

### 6.2. CHRONIC EFFECTS

An Italian study (Costantini and Panella, 1974) reported that alachlor was highly toxic in embryo-larval tests with Bufo vulgaris. Mortality, inhibited development, and histological and enzyme changes were reported in embryos and tadpoles treated at very low levels. Dose levels were not quantified and control data were not available in this abstract (Costantini and Panella, 1974).

## 6.3. PLANT EFFECTS

A Russian abstract (Petrya and Spirescu, 1977) indicates that alachlor inhibits growth, photosynthesis and respiration in the green alga, Chlorella. Chlorophyll synthesis was apparently inhibited at alachlor concentrations >0.0001 mg/ℓ.

## 6.4. RESIDUES

Pertinent data regarding the occurrence of alachlor residues in the aquatic environment or aquatic organisms were not located in the available literature.

## 6.5. OTHER RELEVANT INFORMATION

Additional pertinent data regarding the aquatic toxicity and effects of alachlor on aquatic environments were not located in the available literature.

## 7. EXISTING GUIDELINES AND STANDARDS

### 7.1. HUMAN

According to the Code of Federal Regulations (CFR, 1983), the tolerance for alachlor on raw agricultural commodities intended for human consumption has been established as 0.1 ppm for beans, peas and potatoes and 0.2 ppm for corn under the Federal Food, Drug, and Cosmetic Act (40 CFR 180.249).

In an abstract of a Russian study, Pan'shina (1977) recommends a maximum permissible concentration of alachlor in air of 0.5 mg/m$^3$. The abstract stated that this level was based on rat and rabbit toxicity studies; however, no data were presented.

### 7.2. AQUATIC

Guidelines and standards for the protection of aquatic organisms from the toxic effects of alachlor were not located in the available literature.

## 8. RISK ASSESSMENT

No animal studies have been located in the published literature that assess the carcinogenic potential of alachlor. The results of a number of mutagenicity screening tests do not indicate a substantial probability that alachlor will be found to be carcinogenic.

No information regarding subchronic or chronic toxicity, teratogenicity or other reproductive effects was found in the available published literature.

Statements concerning available literature in this document refer to published, quotable sources and are in no way meant to imply that confidential data, which this document could not address, are not in existence. These data, which are considered Confidential Business Information (CBI), are available in summary form as a CBI Appendix that can be accessed by individuals with appropriate clearance. Conclusions from the CBI Appendix regarding risk assessment, however, can be presented in this chapter.

The CBI Appendix to this document reviews chronic feeding studies that indicate that alachlor is carcinogenic to rats and mice. Alachlor stabilized with 0.5% (w/w) epichlorohydrin was used during the first year of these studies. A $q_1^*$ of $6.9 \times 10^{-3}$ $(mg/kg/day)^{-1}$ was calculated from dose-response data for stomach tumors in female rats fed alachlor in the diet. A more conservative $q_1^*$ of $3.81 \times 10^{-2}$ $(mg/kg/day)^{-1}$ was derived from the dose-response data for nasal turbinate tumors in male rats fed alachlor in the diet. These $q_1^*$ values for humans were calculated using the linearized multistage model developed by Kenneth Crump and adopted by the U.S. EPA (Federal Register, 1980). The conversion factor used to calculate the $q_1^*$ values for humans (assuming 70 kg body weight) from animal data was the cube root of the body weight ratio (Federal Register, 1980).

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## APPENDIX: LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of:

CA SEARCH (Files 308, 309, 310, 311, 320)
TOXLINE
MEDLINE
RTECS
SCI SEARCH
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE

These searches were conducted in May, 1983. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7th), and the following secondary sources were reviewed:


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